SCREENING OF TERM BIRTH ASPHYXIATED INFANTS FOR HEARING LOSS USING OTOACOUSTIC EMISSION

DISSERTATION SUBMITTED FOR

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CERTIFICATE

*This is to certify that the dissertation entitled "**Screening of term birth asphyxiated infants for hearing loss using otoacoustic emission"** submitted by **Dr. P. Jagadeesan** to the Faculty of Paediatrics , The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch VII (Paediatrics) is a bonafide research work carried out by him under our direct supervision and guidance.*

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CONTENTS

INTRODUCTION

INTRODUCTION

Birth asphyxiated infants are prone for multisystem organ damage and hearing impairment is one such thing.

The agony and handicap caused by hearing impairment to a child is far beyond hearing alone, as we all know that a good hearing is essential for normal development of speech, language and cognitive functions of the child. So early diagnosis of hearing impairment is essential for early initiation of rehabilitative measures in a child which is important for future speech, language and cognitive development.

Most of the tests used for assessing the hearing status in a individual requires the cooperation of the subjects, which is obviously not possible in an infant.

In this study we have used Oto acoustic emission test as a screening test for hearing impairment in term birth asphyxiated infants. This is an objective test for hearing impairment, which does not requires the patients cooperation for testing thus can be used effectively in infants.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

White KR, Vohr BR, Maxon AB et al have published a paper in International Journal of pediatric otorhinolaryngology stating that Transient evoked oto acoustic emission is a promising technique for screening newborns for hearing loss and it could be used in a wide basis.

C. Yoshinaga itano, A.L.Gedey & DK Coutler et al have done a study on language development in hearing impaired. Their finding was that in children in whom the hearing loss was identified early ie by six months of age and appropriate rehabilitative measures were started, had a better language scores than those who were identified later than six months of age.[2]

Behrens TR, Vohr BR & White KR et al have published a report quoting the usefulness of Transient evoked oto acoustic emission in universal screening[3] of newborn infants for hearing loss at Rhodes Island.

Kemp DT & Ryans have published a paper quoting the use of Transient evoked oto acoustic emission in neonatal screening programme.[4]

A similar report was also published by Johnson AJ & Maxon AB et al.[5]

Fortnum H, Framworth A & Davis A et al have done a study on the feasibility of evoked oto acoustic emission on inpatient hearing check after meningitis.[6]

Dr. Owen et al from Department of pediatrics Gloucestershire have studied the possibility of community based universal neonatal screening by health visitors[7] using oto acoustic emission. Health visitors were able perform OAE in local health centres. They were able to achieve high population coverage rates.

Welzl Muller K, Boheim K, Stephank et al have published a report on optimizing hearing screening by transient evoked oto acoustic emission in newborn infants.[8] They have adviced the

following. A pass in one ear is enough not required to get pass result in both ears. Perform the testing after the second post partum day. A single testing is not enough and it is a must to perform oto acoustic emission testing atleast twice to minimize the false positive results.

Stevens Jc, Webb HB, Hutchinson J & connell J et al have published a report on comparison between click evoked oto acoustic emission and auditory Brain stem evoked Response[9] which states that the results by both tests are comparable.

Hunter M, Kimml, Cafarelli Dees D et al have published a report stating the feasibility of oto acoustic emission detection followed by Auditory Brain stem evoked response audiometry[10] in universal screening of neonates for hearing impairment.

Heinemann & Bohnert A have published a paper quoting the comparative studies and cost analysis with different instruments in screening for hearing impairment in children[11]. They have suggested that a cost effective way for hearing analysis is to do oto acoustic emission testing universally for all

children and then in those who fail the test Auditory Brain stem evoked response audiometry can be done.

Doyle KJ Burggruff B, Fujikawa S & Kim J have compared the utility of oto acoustic emission testing and auditory brainstem evoked response audiometry[12]. Their inference is that in both the testing modalities there is no obvious difference in test results.

Kennedt CR & Kimml et al have also published a similar report[13] in archives of diseases of child hood.

Alex R. Kemper & Stephen M. Downs et al have done a cost effect analysis of newborn hearing screening strategies comparing the universal screening with oto acoustic emission followed by BERA and Targeted screening of High risk, infants for hearing loss in two stage process [14]. The result of their study was that the universal screening can diagnose more cases at the expense of greater cost and more false positive screening results.

Sun JH, Li J Huang P et al from shanghai medical university [15] have published a report stating that critically ill neonates with some specific high risk factors had a significantly high incidence of hearing impairment and therefore early hearing screening is necessary for neonates who are discharged from Neonatal intensive case unit.

Joint committee on infant hearing have given some guidelines[16] for early defection of hearing impairment. They have adviced hearing screening for infants with.

1. Family History of hereditary childhood sensorineural hearing loss.
2. In utero infections (TORCH, Syphilis)
3. Cranio facial anomalies involving pinna and ear canal.
4. Birth weight less than 1.5kg
5. APGAR scores of 0 to 4 at one minute & 0 to 6 at 5 minutes.
6. Mechanical ventilation lasting 5 days or longer.
7. Hyperbilirubinemia requiring exchange transfusion.

8. Ototoxic medications – multiple courses of aminoglycosides and loop diuretics.
9. Stigmata associated with a syndrome known to include hearing loss.
10. Head trauma associated with loss of consciousness or skull fractures.
11. Bacterial meningitis.
12. Recurrent or persistent otitis media with effusion for atleast 3 months.
13. Parental concern regarding hearing or development delay

American Academy of pediatrics, Task force on newborn infant hearing loss detection and intervention[17] has also proposed similar guidelines for hearing screening.

Christiane Meyer, Jan witte, Agner Hildman et al have published a report on neonatal screening for hearing impairment in which they have considered some other factors[18] also apart from what is stated by Joint committee on infant hearing. They have analysed the relation between hearing loss

and maternal drug abuse, persistent pulmonary hypertension in neonate, intracranial hemorrhage of Grade III and above and periventricular leucomalacia and they have found to have a positive correlation.

Cone Wesson, Barbara Betty & Rsinger et al in their study on identification of neonatal hearing screening have stated that it is essential to do a universal screening [19] rather than a selective high risk screening.

Wessex universal neonatal hearing screening trial group[20] have also adviced universal screening to prevent permanent childhood hearing impairment and its handicaps.

AIM OF THE STUDY

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1. To screen for hearing impairment in term birth asphyxiated hypoxic ischemic encephalopathy stage 2 infants using oto acoustic emission.

2. Early referral of hearing impaired children for rehabilitative measures.

OTO ACOUSTIC EMISSION (OAE)

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Oto acoustic emissions were first discovered by Dr. David Kemp in 1978. The first commercial equipment for recording OAE was produced in USA by 1988. Since then oto acoustic emission testing is used for screening hearing impairment.

What is Oto Acoustic Emission:-

A disturbance in the environment causes sound waves to be created which travel through the air. The sound is funelled into the ear canal by the Pinna and it strikes the tympanic membrane. Then it is transmitted through the middle ear through the ossicles malleus, incus and stapes. The foot plate of stapes conducts the travelling waves across the oval window. Thus the sound reaches the fluid filled cochlea and vibrates the basilar membrane. Each portion of basilar membrane is maximally sensitive to only a limited frequency range. The arrangement is a tonotopic gradient. Regions closest to the oval windows are more sensitive to high frequency stimuli, regions further away are more sensitive to lower frequency stimuli. On the basilar membrane lies the small receptor cells called Hair cells. They are

called so because their appearance resembles small hair follicles. A closer look at hair cells show that they are arranged in rows.

The inner hair cells are arranged in single row and the outer hair cells are arranged in multiple rows. (Three to four)

When the basilar membranes vibrate the hair cells are set into motion and an electro mechanical response is elicited, while an afferent signal is transmitted to the brain an efferent signal is also emitted by the outer hair cells. These efferent signals we call by the name oto acoustic emissions. (OAE). The OAE travels in the reverse direction from cochlea through the ossicular chains vibrating the tympanic membrane to the external auditory canal. When we use special sensitive equipment with a probe in auditory canal these oto acoustic emissions can be recorded.

There are four types of oto acoustic emissions

1. Spontaneous Oto acoustic emission (SOAE)
2. Transient Evoked Oto acoustic emission(TEOAE)

3. Distortion product Oto acoustic emission (DPOAE)
4. Sustained frequency Oto acoustic emission(SFOAE)

Spontaneous Oto acoustic emissions:-

These are sounds produced without any auditory stimuli. These non evoked response usually is measured in narrow bands (< 30 H_z bandwidth) of frequencies. Obtain multiple recordings to ensure replicability and to distinguish the response from the noise floor. SOAE recordings usually span 500 to 7000 Hz frequency range.

Transient Evoked Oto Acoustic emissions:-

In this a auditory stimuli is given and the OAE emitted by outer hair cells are recorded. Clicks are the most commonly used stimuli, although tone burst stimuli may be used. Most commonly 80 to 85 dB SPL stimuli are used clinically. The stimulation rate is less than 60 stimuli per second. TEOAE are generally recorded in the time domain over approximately 20 milli seconds. Alternating responses are stored in alternating computer memory banks A and B. Data that correlate between the two memory banks are considered as a response. Data that

do not correlate are considered noise. When present TEOAE generally occur at frequencies of 500 to 4000 HZ. Data in the time domain then are converted to the frequency domain, usually in octave band analysis.

Distortion product Oto acoustic emissions:-

In this stimuli consists of two pure tones at two frequencies (f_1,f_2 [f_2>f_1]) and two intensity levels (ie L_1 & L_2). The relationship between $L_1 - L_2$ and f_1/ f_2 dictates the frequency response. An f_1/f_2 ratio yields the greatest DPOAE at 1:2 for low and high frequencies and at 1.3 for medium frequencies. To yield an optimal response, set intensities so that L_1 equals or exceeds L_2. Lowering the absolute intensity of the stimulus renders the DPOAE s more sensitive to abnormality. A setting of 65/55 dB SPL L_1/L_2 is frequently used. Responses are usually most robust and recorded at the emitted frequency of $2f_I-f_2$ however, they generally are charted according to f_2 because that region approximates the Cochlear frequency region generating the response.

Sustained frequency oto acoustic emissions:-

SFOAEs are responses recorded to a continuous tone. Because the stimulus and emission overlap in the ear canal, the recording microphone detects both. Therefore interpretation depends on reading a complicated series of rippler in the recording. At present SFOAE s are not used clinically.

In clinical practice TEOAE and DPOAE are most commonly used. In out study we have used TEOAE for screening the infants.

Prerequisites for obtaining oto acoustic emissions:-

1. Un Obstructed outer ear canal (like wax)
2. Hermetic seal of the ear canal with the probe.
3. Optimal positioning of the probe.
4. Absence of middle ear pathology.
5. Functioning Cochlear outer hair cells.
6. A quiscent patient. Excessive movement or vocalization may preclude recording.
7. Relatively Quiet recording environment A sound booth is not required, but a noisy environment may preclude accurate recording.

Nonpathological problems that can cause absence of OAEs

1. Poor probe tip placement or poor seal. Most current equipments alerts clinicians to these problems.
2. Standing waves - most current equipments alerts clinicians to standing waves.
3. Cerumen occluding the canal or blocking a probe port
4. Debris and foreign objects in the outer ear canal.
5. Vernix caseosa in neonates. This is common immediately after birth.
6. Un cooperative patient. Usually, recordings simply are not obtained

Pathological problems that can cause absence of OAEs

1. Stenosis of ear canal.
2. Otitis externa
3. Cysts in ear canal
4. Abnormal middle ear pressure
5. Otitis media
6. Oto sclerosis
7. Middle ear disarticulation
8. Cholesteatoma.

Advantages of OAE:

1. Objective test does not require the cooperation of infants
2. Less time consuming.
3. Less Costly.
4. The probes are less invasive than electrodes required for electrical responses.
5. Can be done in a sleeping child.
6. Less distressing for the parents.
7. All frequencies are tested unlike Brain stem evoked response audiometry
8. Response can be obtained even in the presence of tympanostomy tube.
9. Does not require a sound booth. Can be done in any quiet environment
10. Child needs to be quite and still only for 2 to 5 minutes

Disadvantages:-

1. Cannot be recorded in presence of secretory Otitis media.
2. Requires the child to be completely quiet without noisy breathing or sucking.
3. Can identify only hearing loss more than 30 dB.
4. Gives no indication of the severity of any hearing impairment.

HEARING OR AUDITION

HEARING OR AUDITION

Hearing or audition is the function of ear. Among the special senses inner ear is the first to be fully formed in Humans. It has been proved by various studies that human fetus is able to hear from 27th week of gestation onwards.

Except for the pinna the entire ear is encased in the temporal bone. Anatomically the ear has three parts.

1. External ear is composed of pinna and external auditory canal. Sound waves funnelled by pinna into external auditory meatus. The canal acts as an open pipe resonator.
2. Middle ear composed of Tympanic membrane and three small bones called ossicles viz malleus, incus and stapes. The tympanic vibrates in response to sound waves and this is transmitted through the ossicles to the inner ear. There are two muscles tensor tympani and stapedius which regulate the magnitude of sound.

3. Inner ear consists of cochlea and the vestibule. The vestibule contains the semicircular canals, saccule and utricle. The foot plate of stapes is attached to oval window through which sound is conducted to the inner ear.

Embryology of Ear:-

1. External auditory canal develops from the Ist Branchial groove.
2. Pinna develops from six auditory hillock around the first Branchial grove therefore from Ist and IInd Branchial arches.
3. Middle ear develops from tubotympanic recess from the dorsal part of first pharyngeal pouch.
    a. Tympanic membrane develops from apposition of Tubotympanic recess and Ist Branchial groove.
    b. Malleus and Incus from Ist arch cartilage. Stapes develops from IInd arch cartilage.
4. Membranous Labyrinth from surface ectoderm overlying hindbrain- the otic placode.

Embryological time table

Time in weeks	External & Middle ear	Inner ear
3	First Pharyngeal Pouch	Otic Placode
4	Primitive Meatus	Otic Vesicle
6.	Auditory Hillocks	Endolymphatic Sac & duct
8	Solid epithelial core from primitive meatus towards tympanum	Cartilagenous oocyst
12	Hillocks fuse, ossicles differentiate Tympanic ring ossifies	Organ of corti
16	Ossicles fully formed in cartilage begin to ossify, external ear developed	Fistula antifenestrum appears, ossification of labyrinthine capsule begins.
23	Pneumatisation of upper half of tympanic antrum appears	Ossification of Labyrinthinecapsule nearly complete.
28	Solid Epithelial meatal core canalize	
35	Pneumatisation of cells begin around antrum	
Birth	Pneumatisation accelerates mastoid process appears	
Puberty	Oseous meatus complete Pneumatisation complete except for petrous	

Cochlea:-

Cochlea is buried in hardest bone of the body, the petrous part of temporal bone. Cochlea contains the receptors for hearing. Cochlea is snail shaped. It is 3cm long and makes 2 ¾ turns around the central axis called modiolus. The canal is divided by two membranes i.e. Basilar and Reisners membrane into three compartments, the upper scala vestibuli, the middle scala media, the lower scala tympani scala media contains the endolymph. The scala vestibuli and scala tympani contain the perilymph. The receptors for hearing the hair cells are located on the basilar membrane in scala media.

The hair cells are divided into inner and outer hair cells by pillars of corti. The hair cells contain stereocilia. They are in contact with tectorial membrane. Together they are called as organ of corti. The perilymphatic space of scala tympani is continuous with subarachnoid space of posterior fossa through cochlea aqueduct. This aqueduct is patent in neonatal period. This is the reason for post bacterial meningitic bilateral deafness with vestibular impairment.

Auditory pathway:-

Auditory pathway begins with auditory nerve endings at base of hair cells. The cell bodies of which are in spiral ganglion in Rossenthals canals.

On entering the brain stem auditory fibres bifurcate into upper division and the lower division. The upper division ends in dorsal cochlea nuclei on both sides. So they form a cross over in the midline. This cross over forms the acoustic striae. The lower division ends in ventral cochlear nucleus. Second order neurons from the ventral cochlea nucleus ends in superior olivary nucleus on both sides. This cross over is called by the name trapezoid body.

Second order neurons from dorsal cochlear nucleus ascends in Lateral Lemniscus to relay at the inferior cochlear nucleus.

Similarly fibres ascending from superior olivary nucleus ascend in lateral lemniscus and end in inferior colliculus on enroute some fibres relay in nucleus of lateral lemniscus. There is

a cross over between fibres of Nucleus of lateral lemniscus of both sides which terms the commisure of probst.

There is a cross over of fibres between inferior colliculus on both side which forms the inter collicular commisure.

From the inferior colliculus the fibres ascend to the medial Geniculate body. From the medial geniculate body fibres are projected to the Auditory Cortex (area 41 & 42)

Area 41 the Heschl's gyrus is the primary auditory area where pitch and intensity discrimination occurs. Area 42 is auditory association area where complex synthesis of sound occurs. In Auditory area of brain there is cochleotopic representation as if cochlea is unwinded on cortex with apex represented on outer aspect and base of cochlea on inner aspect.

The two lateral lemniscus and four cross over ie Trapezoid body, Acoustic striae, commisure of probst and inter collicular fibres forms a ladder pattern.

Sound perception involves:-

1. Conduction of sound waves through external, middle and inner ear.
2. Stimulation of receptors (ie) the hair cells of cochlea.
3. Generation of impulse in auditory nerve.
4. Transmission of nerve impulse through auditory pathway.
5. Final processing in cerebral cortex.

Etiology of hearing loss:-

Hearing loss can be central or peripheral in origin. The peripheral hearing loss is further divided into

1. Conductive hearing loss
2. Sensory neural hearing loss
3. Mixed Hearing loss.

1.Conductive hearing loss:-

This is commonly caused by dysfunction in the transmission of sound through the external or middle ear. It may be congenital or acquired.

A. Congenital:-

(i) Anomalies of pinna, external ear canal, tympanic membrane and ossicles. (Most common cause of congenital conductive hearing loss)

(ii) Genetic conditions

a) Pierre Robbin's syndrome

b) Treacher Collins syndrome

c) Klippel-feil syndrome

d) Crouzon's syndrome

(iii) Congenital Cholesteatoma (very rarely)

B. Acquired

(i) Otitis media both acute & chronic variety and its complications like effusion, Cholesteatoma, tympano sclerosis & adhesive otitis.

(ii) Impacted wax or cerumen.

(iii) Impacted foreign body.

(iv) Tympanic membrane perforation (due to trauma or otitis media)

(v) Oto sclerosis.

(vi) Osteogenesis imperfecta

(vii) Osteopetrosis

(viii) Tumors in the ear canal or middle ear (Osteomas, easinophilic granuloma, rhabdomyosarcorna)

2. Sensorineural hearing loss:-

It is the type of hearing loss where the inner ear or the Eighth cranial nerve is involved resulting in impairment of sound perception in the cochlea and higher centre. Sensorineural hearing loss can be because of congenital or acquired causes.

A. Congenital:-

(i) Genetic Causes

(a) Autosommal Recessive syndromes

a. Usher syndrome

b. Pendred syndrome

c. Jervell Nielsen syndrome (a form of the long Q.T interval syndrome)

(b) Autosommal Dominant

a. Waardenburg syndrome

b. Brachio-otorenal syndrome

(c) Sex linked syndrome

a. Alport syndrome

b. Norrie syndrome

(d) Chromosomal Abnormalities:-

a. Downs syndrome

b. Turner's syndrome

c. Trisomy 18 & 13

(ii) Infection (intrauterine infections)

1) Rubella

2) Cytomegalovirus

3) Toxoplasmosis

4) Syphilis

(iii) Teratogenic

1. Thalidomide

2. Quinine

3. Aminoglycosides

4. Loop Diuretics

5. Cisplatin

B. Acquired

1. **Perinatal asphyxia** – very important cause of hearing loss in infants in the absence of any congenital causes of hearing loss
2. **Kernicterus**
3. **Prematurity**
4. **Infections**

a. Bacterial Meningitis

i) Pneumococcus

ii) Hemophilus influenza

iii) Meningococcus

B. Viral Infections

i) Measles

ii) Mumps

iii) Rubella

iv) Varicella

5. **Ototoxic dings:-**

i) Quinine

ii) Aminoglycosides

iii) Loop diuretics

iv) Cisplatin

v) Salicylates.

6. Traumatic Causes

(i) Fracture Temporal bone

(ii) Head injury

(iii) Barotramma

(iv) Noise (acoustic trauma)

Central Causes:-

Auditory deficits originating along the central auditory nervous system pathways from the proximal eighth nerve to the cerebral cortex are generally considered central hearing loss.

Head to foot examination of a case with hearing loss

1. Face and head

Look for any abnormalities in shape, symmetry & presence of any skin tags.

2. Eyes

Look for intercanthal distance, slant, iris colour, vision and retina

3. Ears

- Look for preauricular pits, skin tags, shape of pinna any abnormality in ear canal, patency & size.

- Downward slanting palpebral fissures, coloboma of lower eyelid, malar hypoplasia, malformation of external ear with or without atresia of ear canal, preauricular skin tags, dental malocclusion, teeth hypoplasia & cleft palate are features of Treacher collins syndrome.

- Anterior lenticonus is present in Alports syndome.

- Myopia, cataract, retinal detachment, arthropathy, cleft palate and micrognathia in Sticklers syndrome.

- Retinitis pigmentosa is present in Ushers syndrome

- Bilateral acoustic neuroma, café aulait spots and sub capsular cataract occur neurofibromatosis type 2.

4. Hair

Look for texture, colour & white forelock.

White forelock, premature graying of hair heterochromidia iris, hypertelorism & partial albinism are features of waardenburg syndrome.

5. Neck

- Look for sinus tracts, Thyromegaly
- Thyroid enlargement can occur in Pendreds syndrome.
- Branchial clefts, fistula and cysts with malformed pinna preauricular pits & renal anomalies occur in Branchio otorenal syndrome.

6. Skin

Look for cafeaulait spots, hypopigmentation, hyper-pigmentation and axillary freckling can occur with Neurofibromatosis type I.

7. Balance & gait

Gait disturbance can occur in Ushers syndrome due to vestibular dysfunction

MATERIALS & METHODS

MATERIALS & METHODS

Study design

Prospective longitudinal study

Study population

Term birth asphyxiated Hypoxic ischemic encephalopathy stage 2 infants attending the well baby clinic in Institute of Child Health and Research Centre in Government Rajaji Hospital attached to Madurai Medical College.

Study period

From August 2004 to January 2006.

Inclusion Criteria:-

2. Term babies
3. Birth asphyxiated HIE stage 2 infants.
4. With normal developmental milestones.
5. Without severe neurologic impairment

Exclusion Criteria:-

1. Preterm babies.
2. Babies with severe neurological impairment.
3. Babies with other risk factors like Hyperbilirubinemia
4. Babies with other congenital anomalies.
5. Babies with family history of hearing loss.
6. Very low birth weight babies.

Method:

Term birth asphyxiated infants who are on regular follow up are initially screened for hearing by response to turning to ring of a Bell at around 6 months of age. The six month cut off is taken because the average time when a child turns to sound is around 5.8 months according to Trivandrum developmental screening test. Those children who have doubtful turning to sound by ring of a bell are subjected to oto acoustic emission test after parental consent which is an objective test for hearing impairment.

RESULTS AND OBSERVATIONS

RESULTS AND OBSERVATIONS

A Table 1

Follow up of children enrolled

	Children	
	No	**%**
Children followed up	176	62.4
Children lost to follow up	106	37.6
Total Children enrolled	282	100

Out of the 282 children enrolled for the study, 37.6% of the children were lost to follow up due to various reasons.

Table 2

Bell test

Bell test result	Children	
	No	**%**
Children found normal	128	72.7
Children suspected to be defective	48	27.3
Total children followed up	176	100

Among the 176 children followed up, 48 (27.3%) children were suspected of having defect in the Bell test.

Table 3: Bell test and OAE test.

OAE Result	Children	
	No	**%**
Children confirmed defective as per OAE	8	16.7
Children confirmed normal as per OAE	40	83.3
Total Children suspected of having hearing defect as per Bell test	48	100

OAE test confirmed hearing defect in 16.7% of the cases among children suspected of having hearing defect in Bell test.

Table: 4 Sex wise distribution

Sex	Hearing impairment Present		Hearing impairment absent	
	No	%	No	%
Male	6	17.1	29	82.9
Female	2	15.4	11	84.6
Total	8	20	40	80

p= 0.6266

There was no statistically significant difference in incidence of hearing defects among birth asphyxiated male & female babies.

Table 5 Obstetric history

Obstetric History	Hearing impairment Present		Hearing impairment Absent	
	No	%	No	%
B.O.H. (n=10)	2	20	8	80
Normal (n=38)	6	15.8	32	84.2
Total (n=48)	8	20	40	80

P=0.5359

The percentage of hearing defect was slightly more among those with previous bad obstetric history. But it was statistically not significant.

Table 6: Type of delivery

Type of delivery	Hearing impairment present		Hearing impairment absent	
	No	%	No	%
Normal Delivery (n=32)	4	12.5	28	87.5
Assisted / LSCS (n=16)	4	25	12	75
Total(n=48)	8	20	40	80

p = 0.2424

The percentage of hearing loss among birth asphyxiated infants delivered by assisted/LSCS delivery was twice that of those delivered by Labour natural.

Table 7 : Apgar score

Apgar Score 1'	Hearing impairment present		Hearing impairment absent	
	No	%	No	%
< 4 (n=11)	4	36.4	7	63.6
> 4 (n=37)	4	10.8	33	89.2
Total (n=38)	8	20	40	80
p = 0.0482				

Apgar Score 5'	Hearing impairment present		Hearing impairment absent	
	No	%	No	%
< 4 (n=10)	4	40	6	60
> 4 (n=38)	4	10.5	34	89.5
Total (n=48)	8	20	40	80
p = 0.0471				

There is a significant correlation between APGAR score and hearing defect. When the APGAR score was less than 4 at 1 or 5 minutes. (ie those with severe birth asphyxia) incidence of hearing defects increases significantly.

Table 8 Birth Weight

Birth Weight	Hearing impairment present		Hearing impairment absent	
	No	%	No	%
< 2.5 Kg(n=6)	1	16.7	5	83.3
2.5 – 3.0 Kg (n=34)	6	17.6	28	82.4
> 3.0 Kg (n=8)	1	12.5	7	87.5
Total	8	20	40	80

p=0.6872

Very low birth weight infants have been excluded from the study. In the study group there was no obvious difference in incidence of hearing defect in various weight groups.

Table 9 Neurosonogram /CT Brain Results

Neuro Sonogram/CT Brain result	Hearing impairment present		Hearing impairment absent	
	No	%	No	%
Normal (n=36)	5	13.9	31	86.1
Abnormal (n=12)	3	25	9	75.0
Total	8	20	40	80

P=0.3137

The percentage of hearing loss was high among those with abnormal neurosonogram/ CT finding when compared to those with normal findings.

Table 10 Duration of Hospitalisation

Duration of Hospitalisation (in days)	Hearing impairment present		Hearing impairment absent	
	No	%	No	%
< 5 days (n=5)	-	-	5	100
5-10 (n=28)	4	14.3	24	65.7
> 10 (n=15)	4	26.7	11	73.3
Total (n=48)	8	20	40	80

P=0.4034

In those with less than 5days hospitalization there were no hearing defect & the percentage of hearing defect was more in those with more than 10 days hospitalization than those with less than 10 days hospitalization.

Table 11 Socio economic Status

Socio economic status	Hearing impairment present		Hearing impairment absent	
	No	%	NO	%
I (n=0)	-	-	-	-
II (n=0)	-	-	-	-
III (n=9)	-	-	9	100
IV (n=14)	2	14.3	12	85.7
V (n=25)	6	24	19	76
Total (n=48)	8	20	40	80

P=0.1509

The percentage of hearing defect was more in these with class V socio economic status than there with class IV socio economic status. No cases was reported in class III socio economic status.

Discussion

Discussion

In out study we have screened all term birth asphyxiated HIE state II infants for hearing loss using a bell and if they are

found to have doubtful turning to sound in bell test they were subjected to oto acoustic emission testing. Since the mean age of turning to sound is around 5.8 months we have taken 6 month as cut off point and screened all infants at 6th months while they are on follow up.

A total of 282 cases of term birth asphyxiated HIE state II infants were registered for study and out of which 106 cases were lost to follow up for various reasons. Of the remaining 176 cases who were on regular follow up 48 infants had doubtful turning to sound when they were tested by bell method. Of these 48 cases 35 were males 13 were females. These 48 cases were subjected to screening by oto acoustic emission testing.

Of the 48 cases tested by oto acoustic emission 40 infants passed the test and the remaining 8 cases did not pass the test. Of these 8 cases 6 were males and 2 were females. So there is no obvious difference in incidence of hearing loss in birth asphyxiated infants in both sexes.

Of the 8 cases mothers of 2 cases had previous bad obstetric history and in the remaining 6 cases the obstetric history was normal so previous had obstetric history does not affect the out come of hearing significantly.

When comparing the hearing outcome in various mode of delivery we could find that the percentage of infants with hearing impairment in those with assisted delivery was twice as compared to babies delivered by labour natural. But the confounding factor here in that in cases which required assisted delivery already they were in a state of prolonged labour which may itself contribute to perinatal asphyxia.

Very low birth weight infants have been excluded from the study. In this study group were the birth weight ranged from 2.0kg to 3.5 kg there was no obvious significant difference in incidence in any particular weight categories of infants.

As this study was conducted in a Government hospital settings only cases belonging to class III, class IV & class V socio economic status scaling of Kuppuswamy who have utilized the hospital services have been included in the study. So the

incidence of hearing impairment could not be assessed in all social classes. But among this cases no infants was found to be hearing impaired in class III and the percentage of hearing impairment was slightly higher in those belonging to class V when compared to class IV socio economic strata. But a conclusion cannot be reached on this point as this is not a population based study and most of the cases attending the government hospital belonged to lower socio economic strata.

There was a significant correlation between APGAR score and the incidence of hearing impairment. The incidence of hearing impairment was significantly higher in those infants with severe birth asphyxia i.e. infants with 5 minute APGAR score of less than 4 when compared with those of APGAR score of more than 4 at 5 minutes. So the incidence of hearing impairment is directly proportional to the severity of asphyxia.

The percentage of infants with hearing loss was higher in those with abnormal findings in neurosonogram or CT Brain when compared to those with normal neurosonogram or CT Brain findings but the P value was not significant. So this abnormal

neuro imaging finding can not be taken as a positive collaborative evidence.

When considering the duration of hospitalization and number of infants with hearing loss the following observations were made. There was no infant with hearing impairment in the group of infants with less than five days of hospitalisation. But the percentage of infants with hearing impairment was twice in those group of infants who required more than 10 days of hospitalisation when compared to those group of infants with less than 10 days of hospitalisation. So it is obvious that those infants with prolonged convulsions who required longer duration of hospital stay to control convulsions had greater incidence of hearing impairment.

LIMITATIONS

LIMITATIONS

1. Oto acoustic emission was not done for all cases.

2. The testing was done only once and was not repeated.

3. All infants in NICU have got aminoglycosides the effect of which could not be ruled out.

CONCLUSION

CONCLUSION

1. Birth Asphyxia can cause hearing impairment in infants.
2. The incidence of hearing impairment is directly proportional to the severity of asphyxia.
3. The incidence of hearing impairment is more in those who required longer duration of inpatient care for control of seizures.
4. The incidence of hearing impairment is higher in those who required assistance during delivery than those who were delivered by labour natural.
5. Screening for hearing impairment is essential in all high risk infants.

RECOMMENDATIONS

RECOMMENDATIONS

1. Universal screening of hearing impairment is essential in all newborns as this can detect hearing impairment at an early stage and early referral for rehabilitation.

2. If not possible because of financial constraints atleast all high risk infants have to be screened for hearing impairment following discharge from neonatal intensive care unit.

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BIBLIOGRAPHY

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PROFORMA

PROFORMA

Screening of term birth asphyxiated infants for hearing loss using oto acoustic emission

Name :

Age : Sex :

Mother : Father :

Address :

Date of Admission :

Date of discharge :

O/P No. :

Family History :

Consanguinity

Other Siblings

Family History of hearing loss

Antenatal history :

H/O exanthematous fever

H/O drug intake

H/O radiation exposure

Natal & Postnatal History :

Mode of Delivery Birth weight

Gestational age

H/O Birth asphyxia

Apgar 1’

5’

H/O Neonatal convulsions

H/O Neonatal Hyperbilirubinemia

H/O Hospitalisation

H/O Seizures & Treatment

Developmental History :

Socio Economic History :

General Examination :

Alertness

Neurocutaneous markers

Abnormal Facies

Developmental anomalies

Vitals :

HR

RR

CRT

Weight

Height

Head circumference

CNS :

Consciousness

AF

Pupils

EOM

Facial Nerve

Response to Sound :

Turning to bell

Startle response

Nasal regurgitation

	R	L
Tone		
UL		
LL		
Power		
UL		
LL		
DTR		
Plantar		
ATNR		

CVS :

S1 S2

Murmur

RS

Trachea

Air Entry

Breath sounds

Abdomen

Soft

Organomegaly

INVESTIGATION

Hb

TC

DC

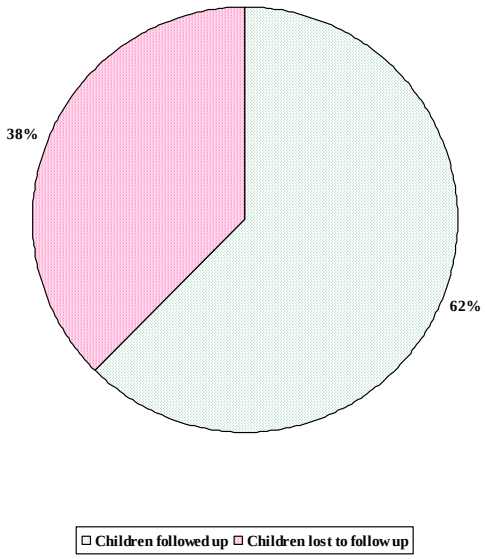
EEG

Neurosonogram/CT Brain

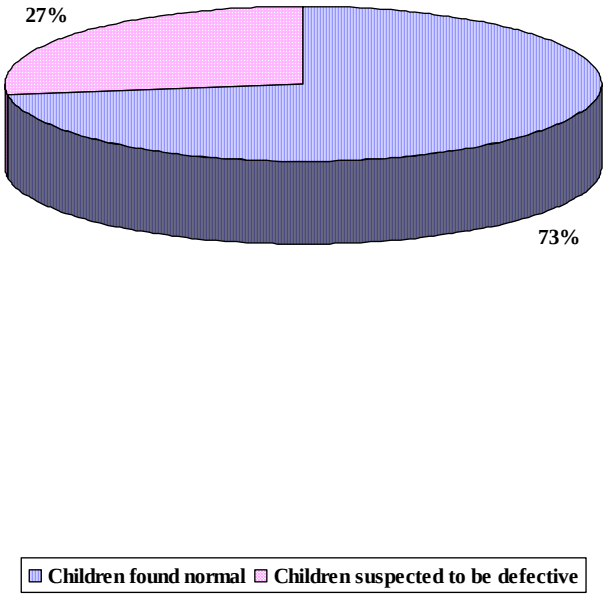
Oto acoustic emission

Inference

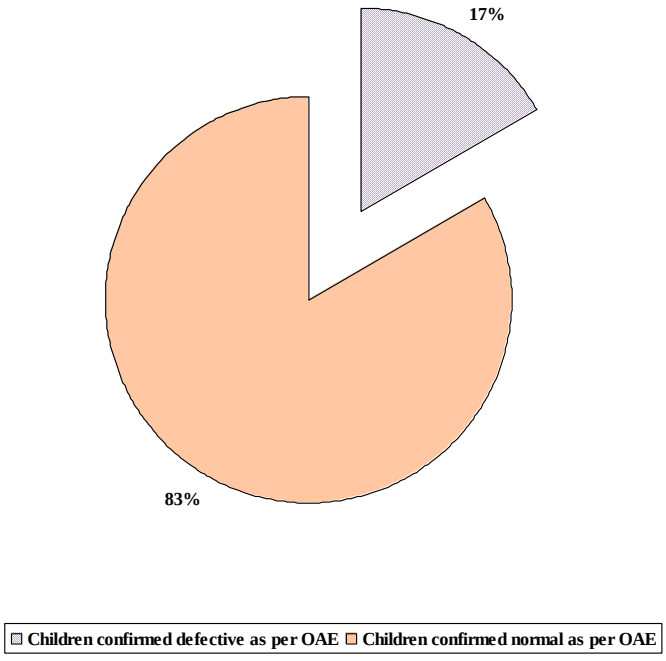
Follow up of children enrolled



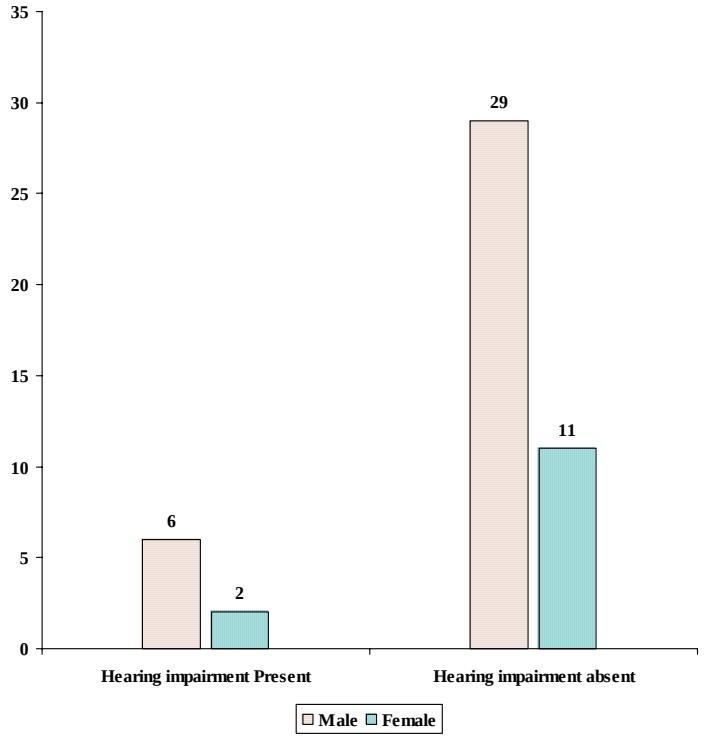
Bell Test


27%
73%
Children found normal
Children suspected to be defective

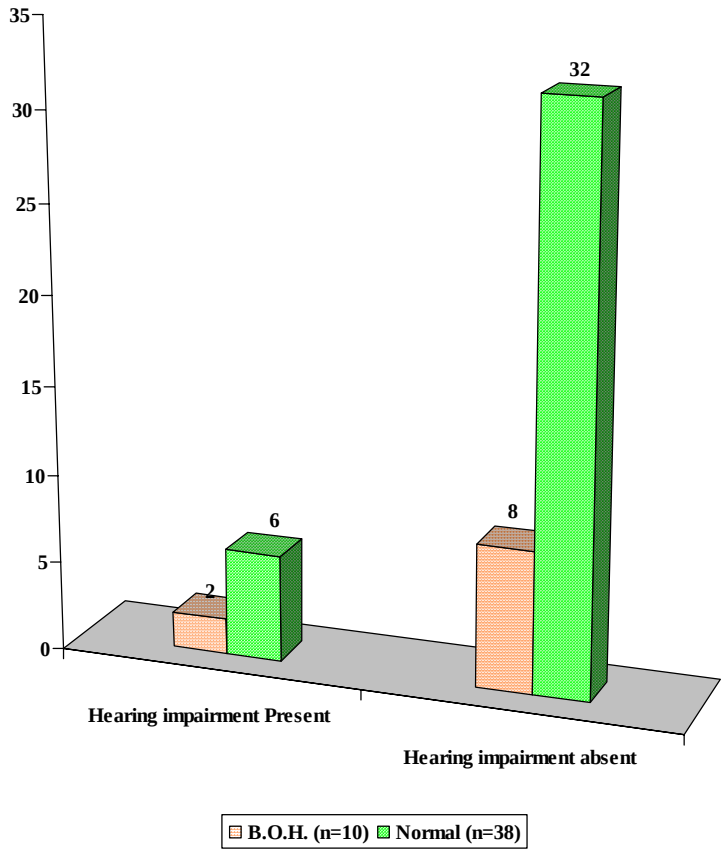
Bell Test and OAE Test



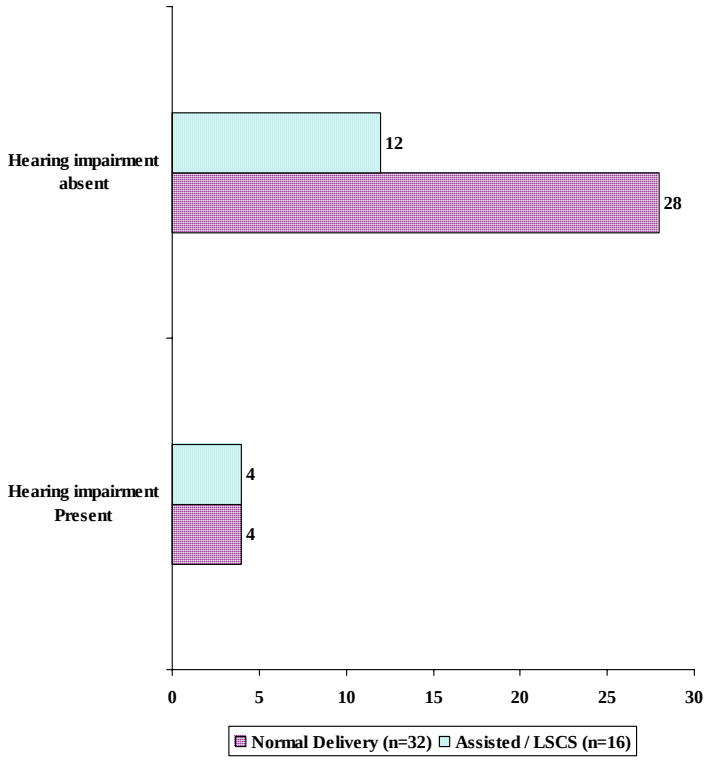
Sex wise distribution



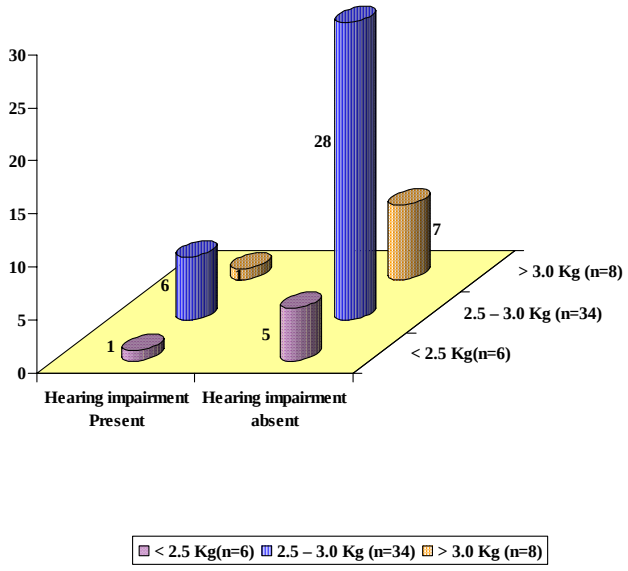
Obstetric History



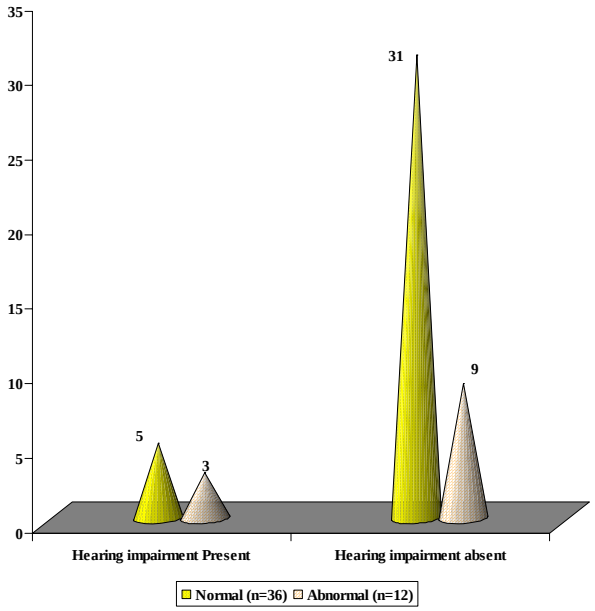
Type of Delivery



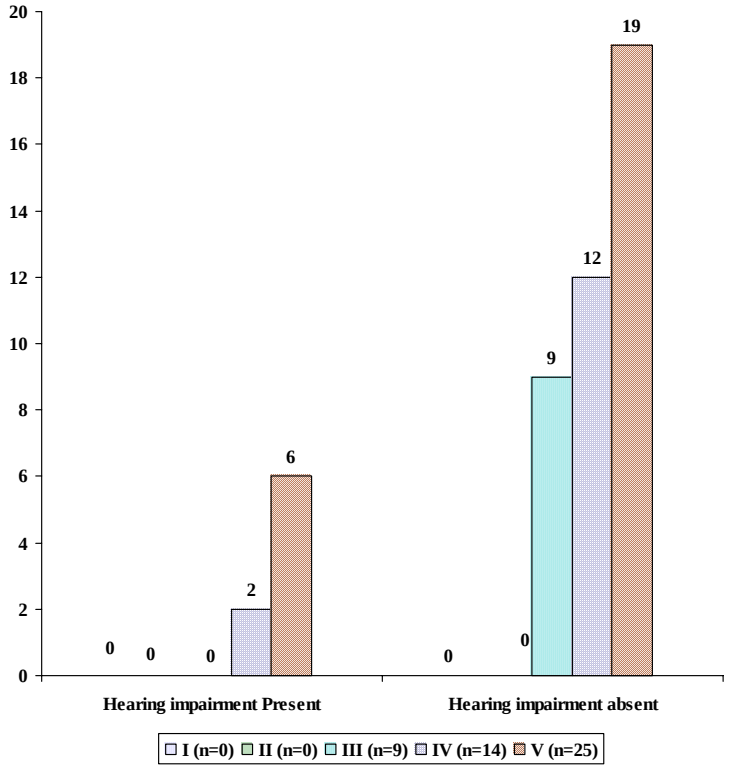
Birth Weight



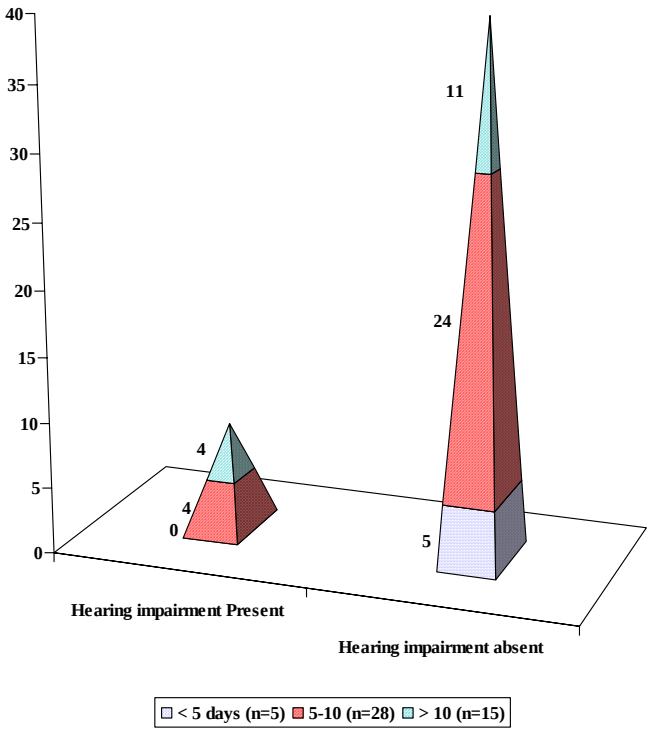
Neuro Sonogram/CT Brain result



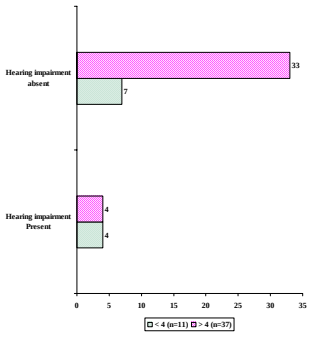
Socio Economic Status



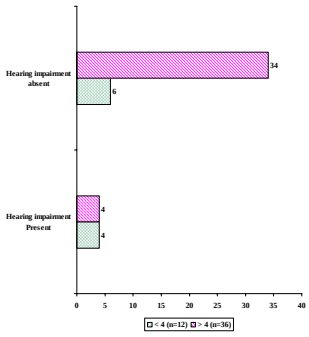
Duration of Hospitalisation



Apgar Score 1'



Apgar Score 5'



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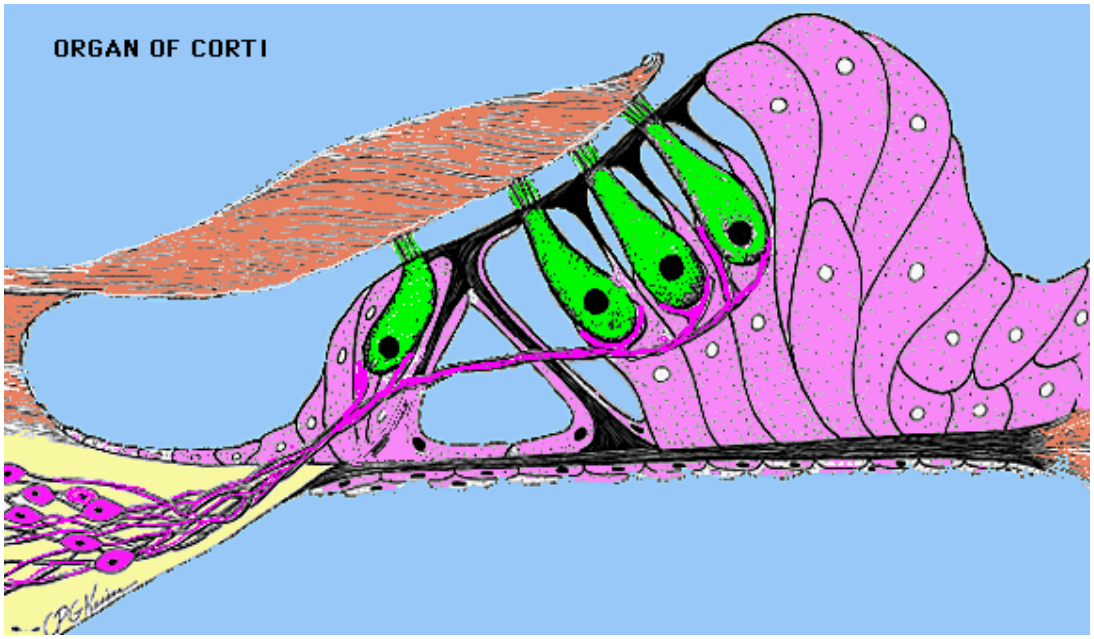
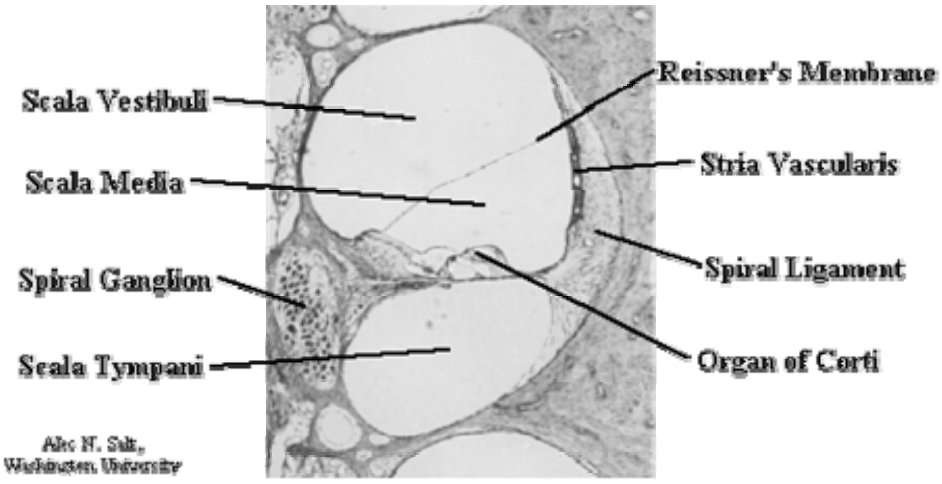
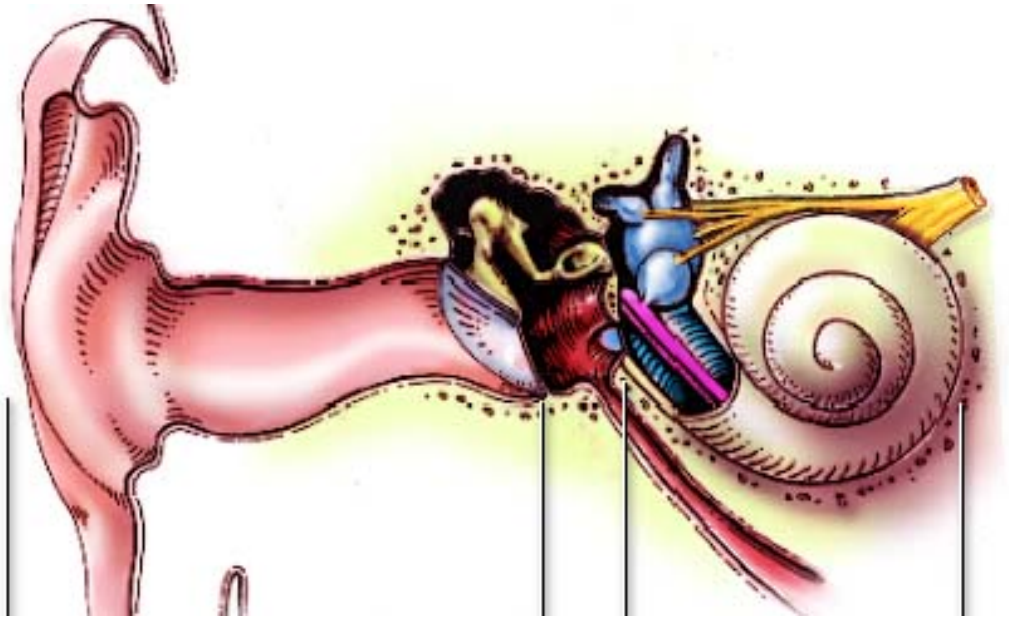


COCHLEAR CUT SECTION



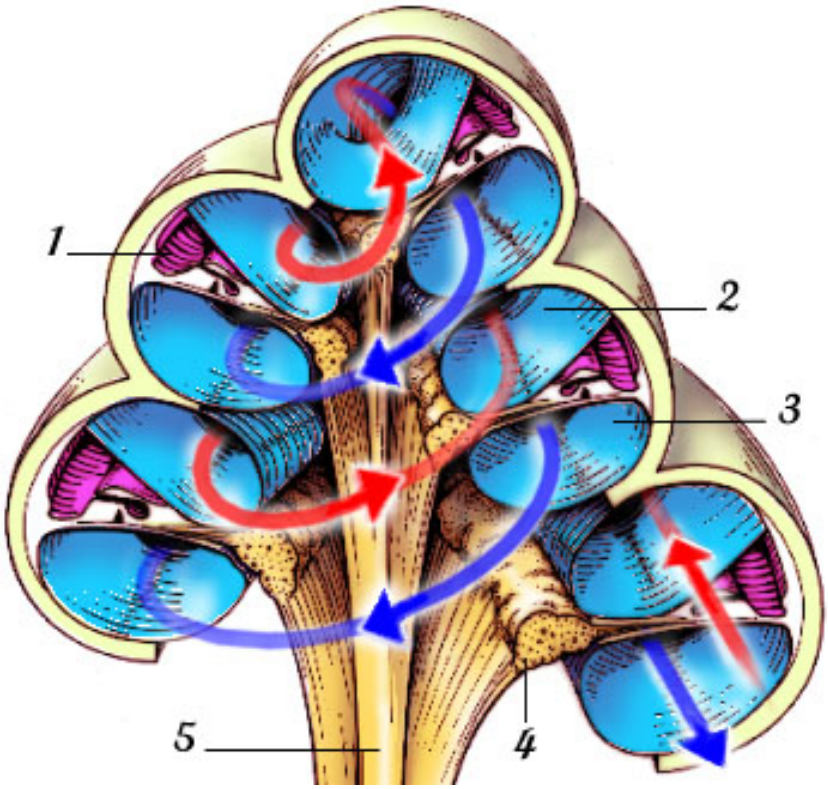
EAR



OTO ACOUSTIC EMISSION EQUIPMENT

OAE ROOM

SOUND TRANSMISSION IN COCHLEA



OAE TEST RESULT SHOWING FAIL IN THE TEST

MRNO 2272
Date 17.5.05

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TE 64 sec avg V7.62

NAME: Mani Selvam 8 months

F	P	TE	NF	SN
1.5	83	-11	-9	-2
2.0		-22	-18	-5
2.5		-23	-24	1
3.0		-31	-25	-6
3.5		-22	-26	4
4.0		-26	-21	-6

F ┼─┼─┼─┼─┼─┼
1.5
2.0
2.5
3.0
3.5
4.0
F ┼─┼─┼─┼─┼─┼
-15 -10 -5 0 5 10
Level (dB) ▒-NF ▪-TE

Left : Refer

OtoRead

OTOACOUSTIC EMISSIONS TEST

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TE 64 sec avg V7.62

NAME:.....

F	P	TE	NF	SN
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2.0		-20	-15	-4
2.5		-26	-23	-3
3.0		-24	-25	1
3.5		-26	-22	-4
4.0		-24	-21	-2

F ┼─┼─┼─┼─┼─┼
1.5
2.0
2.5
3.0
3.5
4.0
F ┼─┼─┼─┼─┼─┼
-15 -10 -5 0 5 10
Level (dB) ▒-NF ▪-TE

Right : Refer

OAE TEST RESULT SHOWING PASS IN THE TEST

MRNO : 2451.

??????????OtoRead"

OTOACOUSTIC EMISSIONS TEST

Left 16-Jun-05 01:55 PM
TE 64 sec avg V7.62

NAME: M. sthemadharshini

F	P	TE	NF	SN	
1.5	83	3	12	-9	
2.0		1	1	0	
2.5		0	-5	4	P
3.0		-8	-7	-1	
3.5		-10	-14	5	P
4.0		-7	-16	9	P

Left : Pass

OtoRead

OTOACOUSTIC EMISSIONS TEST

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TE 43 sec avg V7.62

NAME:.....

F	P	TE	NF	SN	
1.5	83	7	5	2	
2.0		6	0	6	P
2.5		-4	-6	1	
3.0		-7	-17	9	P
3.5		-5	-14	9	P
4.0		-9	-10	1	

Right : Pass

MASTER CHART

Name	OP No.	Sex	BOH	LN/Assisted or LSCS	B.W	APGAR		Duration of Hospitalisation	Socio economic Status	Neur Sonogr / CT Br
						1’	5’			
B/o Jeyanthi	308/04	M	B	A	2.5	3	4	12	V	A
'O Mariam Beevi	327/04	F	N	LN	3.1	5	6	4	III	N
) Mahabaha Beevi	332/04	M	B	A	2.5	3	4	8	IV	A
B/o Prema	356/04	M	N	LN	2.6	5	6	7	IV	N
B/o Shanthi	360/04	M	N	A	2.3	5	6	8	III	N
3/o Vijayalaxmi	376/04	M	N	LN	2.8	4	5	4	IV	N
B/o Shanthi	384/04	F	N	A	2.6	3	3	9	V	N
B/o Poochendu	401/04	M	N	LN	2.7	5	6	9	V	N
Syed Ali Fathima	412/04	M	N	LN	2.4	5	6	6	III	N
B/o Venila	419/04	M	N	LN	2.5	4	5	8	IV	N
B/o Ramalaxmi	428/04	M	B	A	2.6	4	4	14	V	A
B/o Raja laxmi	442/04	M	N	LN	2.8	3	3	12	V	N
'o Karthigai Rani	463/04	F	N	A	2.9	5	6	7	IV	N
B/o Rani	466/04	M	N	LN	3.3	4	5	9	V	N
B/o Rakku	475/04	M	N	LN	2.7	5	6	4	III	N
B/o Shanthi	505/04	M	N	LN	2.3	5	6	9	V	N
3/o Muthulaxmi	537/04	M	N	LN	2.8	5	6	8	IV	N
3/o Muneeswari	553/04	M	N	LN	2.9	4	6	9	V	N
B/o Chitra	568/04	M	N	A	2.7	3	3	14	V	A
B/o Jeyanthi	8/05	F	B	A	2.6	3	4	13	V	A
B/o Ashwarya	18/05	M	N	LN	3.1	6	7	7	V	N

Name	OP No.	Sex	BOH	LN/Assisted or LSCS	B.W	APGAR		Duration of Hospitalisation	Socio economic Status	Neur Sonogr / CT Br
						1’	5’			
B/o Laxmi	23/05	F	N	LN	2.9	5	6	12	IV	N
B/o Sameema	29/05	M	B	A	2.4	4	5	14	IV	N
B/o Sasikala	38/05	M	N	LN	3.1	6	6	9	III	N
B/o Thiravium	43/05	F	N	LN	2.5	5	6	9	IV	N
B/o Puspham	54/05	M	N	LN	2.8	4	5	7	V	N
3/o Backialaxmi	62/05	M	B	LN	2.8	3	4	13	V	N
B/o Sakeela	76/05	M	B	A	2.7	5	6	12	V	A
B/o Mayil	85/05	F	N	LN	2.3	5	6	8	IV	N
B/o Punitha	93/05	M	N	A	2.6	5	6	8	V	N
3/o Vijayalaxmi	101/05	M	N	A	3.1	5	5	14	III	N
B/o Nafeesa	130/05	M	N	LN	2.5	4	5	6	V	N
/o Regina Dhoni	146/05	F	N	LN	2.5	5	6	4	IV	N
/o Azhagumeena	155/05	F	N	LN	2.9	3	3	8	V	N
B/o Deepa	162/05	M	N	LN	2.7	4	6	9	V	N
B/o Alamely	171/05	M	N	LN	3.2	4	6	8	III	N
B/o Amutha	178/05	M	N	LN	2.8	5	6	13	V	N
B/o Laxmi	193/05	M	N	LN	2.9	4	5	8	IV	N
B/o Mahadevi	209/05	F	B	A	2.5	3	4	7	V	A
/o Kurinji malar	220/05	M	B	LN	2.3	5	6	4	V	A
B/o Paranjothi	225/05	M	N	A	2.5	3	4	12	V	A
/o Ananda Valli	237/05	M	N	LN	2.5	5	6	6	IV	N
B/o Suba	250/05	F	N	LN	2.8	4	5	4	III	N
Panchavarnam	265/05	F	N	A	2.6	3	5	14	V	A
B/o Nagalaxmi	277/05	M	N	LN	3.2	4	4	9	IV	N

Name	OP No.	Sex	BOH	LN/Assisted or LSCS	B.W	APGAR		Duration of Hospitalisation	Socio economic Status	Neur Sonogr / CT Br
						1’	5’			
B/o Kumutha	298/05	F	N	LN	3.1	5	6	8	III	N
B/o Rekha	317/05	M	B	A	2.8	3	4	13	V	A
B/o Rajeswari	349/05	M	N	LN	2.5	4	4	8	V	A